

ANONYMOUS

Witness name:

GRO-B

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Dated: 11th August 2022

INFECTED BLOOD INQUIRY

EXHIBIT WITN2151024

Date 1 April 1997
 Our Ref JA/SF
 Your Ref
 Ext No

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GRO-B

GRO-B

I refer to my letter of 20th March. I now attach copy report and correspondence between Brian Donald of the Solicitors Hepatitis Group and Professor Zuckerman, for your information.

You will recall that the Scottish Group were hoping to obtain copies of medical reports and Counsels' opinion obtained by their English equivalent, and as soon as I hear anything on this front, I will be in touch again.

Yours sincerely,

GRO-C

Jean Abbot

Enc.

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From THE DEAN

PROFESSOR ARIE J. ZUCKERMAN, MD, DSc, FRCP, FRCPath

Aspects of Viral Hepatitis Type B and C

A Report prepared for the Solicitors' Hepatitis Group (Scotland)

June 1996

1. Introduction

Viral hepatitis is a major public health problem throughout the world affecting several hundreds of millions of people. Viral hepatitis is a cause of considerable morbidity and mortality in the human population both from acute infection and chronic sequelae which include, with hepatitis B and hepatitis C infection, chronic active hepatitis, cirrhosis and primary liver cancer.

The hepatitis viruses include a range of unrelated and often unusual human pathogens:

Hepatitis A Virus (HAV), a small unenveloped symmetrical RNA virus which shares many of the characteristics of the picornavirus family. This virus has been classified as hepatovirus within the heparnavirus genus and is the cause of infectious or epidemic hepatitis transmitted by the faecal-oral route.

Hepatitis B Virus (HBV), a member of the hepadnavirus group, double-stranded DNA viruses which replicate by reverse transcription. Hepatitis B virus is endemic in the human population and hyperendemic in many parts of the world and is transmissible by blood, blood to blood contact and by the sexual route. Natural hepadnavirus infections also occur in other mammals including woodchucks, beechy ground squirrels and ducks.

Hepatitis C Virus (HCV), an enveloped single-stranded RNA virus which appears to be distantly related (possibly in its evolution) to flaviviruses, although hepatitis C is not transmitted by arthropod vectors. Several genotypes have been identified. Infection with this virus is common in many countries, and it is associated with chronic liver disease and also with primary liver cancer at least in some countries.

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Hepatitis D Virus (HDV) is an unusual single-stranded circular RNA virus with a number of similarities to certain plant viral satellites and viroids. This virus requires hepadnavirus helper functions for propagation in hepatocytes, and is an important cause of acute and severe chronic liver damage in some regions of the world.

Hepatitis E Virus (HEV), is an enterically-transmitted non-enveloped, single-stranded RNA virus, which shares many biophysical and biochemical features with caliciviruses. Hepatitis E virus is an important cause of large epidemics of acute hepatitis in the subcontinent of India, Central and South-East Asia, the Middle East, parts of Africa and elsewhere; and this virus is responsible for high mortality during the third trimester of pregnancy.

The GB Hepatitis Viruses (GBV-A, GBV-B and GBV-C:HGV).

The GB hepatitis viruses were cloned recently and preliminary genomic characterisation shows that they are related to other positive-stranded RNA viruses with local regions of sequence identity with various flaviviruses.

Phylogenetic analysis of genomic sequences showed that these viruses are not genotypes of hepatitis C virus. The hepatitis G virus (HGV) which was cloned independently, is believed to be very similar to if not identical with GBV-C.

Viral hepatitis (and more recently HIV) constitute the main hazard of the transfusion of blood and certain plasma components. Several types of viral hepatitis are recognised now, as outlined above. The availability of specific serological tests for hepatitis A and hepatitis B revealed that hepatitis A is not transmitted by blood and blood products and provided evidence for the existence of other hepatitis viruses.

2. * **Hepatitis B and Blood Products** *

* A WHO Committee noted in 1974 (WHO Technical Report Series No 570, Geneva, 1975) that rates of hepatitis B surface antigen positivity vary markedly among different donor populations, and according to the sensitivity of the test used for detection. • It was also recorded that in general, paid or commercial blood donors in the USA have been found to carry the antigen 5-10 times more frequently than volunteer donors. *

A discouraging finding in prospective studies of transfused patients was that hepatitis cases continued to occur even in recipients of blood which was non-reactive for hepatitis B surface antigen by the most sensitive radioimmunoassay technique available at the time. It became apparent that although some recipients had hepatitis B, it appeared that many had infections that were not caused by hepatitis B virus.

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Two ways of improving the safety of high-risk plasma derivatives were considered: 1) The use of the most sensitive test for hepatitis B surface antigen for every unit of plasma for fractionation; and 2) The development of methods to remove selectively or to inactivate residual virus.

The WHO Expert Committee on Hepatitis, 1976 (Technical Report Series No 602, Geneva, 1977) recommended that since many studies have shown ~~that~~ paid donors constitute a particularly high-risk group for transmitting hepatitis, every effort should be made to introduce an entirely voluntary blood donor system.

In 1979, an informal consultation on viral hepatitis was held by WHO (McCollum R and Zuckerman AJ. Journal of Medical Virology, 1981; 8: 1-29). It was noted that hepatitis B as well as non-A, non-B hepatitis transmitted by high-risk donors remain a significant problem and such donors should be identified, other than by specific serological testing (not available at the time for non-A, non-B hepatitis). It was also recommended that Government Health Authorities should control trade in commercially acquired blood and blood derivatives in order to reduce the transmission of hepatitis virus infections.

Note that the Federal Government of the USA required that all blood donor units be clearly designated as "paid" or "volunteer"; Federal Register, 1978.

3. Risk of Hepatitis after Plasma Concentrates of Commercial or Volunteer Origin

Surveys and clinical studies of post-transfusion hepatitis in the UK indicated that the incidence of hepatitis after the use of blood products obtained from voluntary donors was 2-4%. Therefore, even on the basis of lower reported attack rates in the UK e.g. 2.4% after a mean exposure to 7.4 units of blood products, it became virtually certain that a susceptible patient exposed to more than 300 donor units of blood products will develop hepatitis. Therefore, the overall attack rates approached 100%, whether concentrates were of commercial or volunteer origin.

The introduction of viral inactivation by heating of clotting factor concentrates in 1985-1986 has been largely effective.

4. Non-A, Non-B Hepatitis: Hepatitis C

As specific diagnosis of the different types of viral hepatitis became possible some 20 years ago it became apparent that there was one form, previously unrecognised and unrelated to hepatitis A, B, or D, that was transmitted principally by the parenteral route. This became known as non-A, non-B hepatitis.

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Results obtained from several carefully conducted surveys of post-transfusion hepatitis in the United States and elsewhere provided strong epidemiological evidence of "guilt by association". This form of non-A, non-B hepatitis has been found in every country in which it has been sought; and it shares several features with hepatitis B. In countries where all blood donations are screened for hepatitis B surface antigen by very sensitive techniques non-A, non-B hepatitis may account for as many as 90% of all cases of post-transfusion hepatitis. Outbreaks of non-A, non-B hepatitis have also been reported after treatment with blood clotting factors VIII and IX. Non-A, non-B hepatitis has occurred in haemodialysis and other specialist units, among drug addicts, and after accidental inoculation with contaminated needles and other sharp objects. Mother to infant transmission has been reported. In Britain and in several other countries some cases are not associated with transfusion, and such sporadic cases of non-A, non-B hepatitis account for 10-25% of all adult patients with clinical viral hepatitis. The route of infection or the source of infection cannot be identified in many of these patients.

Although in general the illness is mild and often subclinical or anicteric, severe hepatitis with jaundice does occur, and non-A, non-B hepatitis accounts for a substantial fraction of all fulminant hepatitis. In many patients the infection may be followed by prolonged viraemia and a persistent carrier state. Studies of the histopathological sequels of acute non-A, non-B hepatitis infection showed that chronic liver damage, which may be severe, may occur in as many as 40-50% of patients.

* A News Release by the Chiron Corporation in May 1988 reported the identification and cloning of hepatitis C virus (Wall Street Journal, 11 May 1988), but publication in the scientific literature was delayed until the following year (Choo Q-L et al. Science, 1989; 244: 359-362). *

There is no doubt that the ability to detect antibody to hepatitis C virus based on the technology introduced by the Chiron Corporation in 1989, generally several weeks or months after acute infection was an important advance and a useful screening procedure. The assay was based at the time on anti-C-100-3 antibody. C100 is located within a small non-structural region (363 amino acids) of the polyprotein of hepatitis C virus. This region of the viral polyprotein was recreated by the ligation of four overlapping clones. There were, however, a number of problems:

1. Some individuals infected with hepatitis C virus do not mount a detectable antibody response to C100-3. Further, the "window phase" i.e. from the time of infection to the appearance of antibody may vary from weeks to many months.
2. There were a large number of false positive and false negative reactions. The number of false reactions increased even further when the test was applied to frozen and thawed samples.

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3. The lack, at the time, of confirmatory assays for repeatedly reactive or borderline (indeterminant) reactions.

Nevertheless, it was generally agreed that testing for hepatitis C should be introduced as soon as it was practical.

Practical considerations included the following:

1. Evaluation of the tests;
2. Pilot studies;
3. Availability of additional equipment;
4. Training of technical personnel and provision of scientific information to staff and medical advice to donors;
5. Securing capital and recurrent funding at a time of severe financial constraints.

Preliminary trials were completed in the Autumn of 1990, but at about the same time the only two manufacturers, the Chiron Corporation and Abbott Laboratories both from the USA, were planning to introduce new, more sensitive and specific second generation tests. The second generation tests became available three months later (February 1991) and required evaluation. Further evaluations were undertaken and when completed it became apparent that one manufacturer provided a pre-production batch. The final test format was different from the pre-production batch and all the tests had to be repeated in the Spring and early Summer of 1991.

Testing was introduced throughout the UK at a common date on 1st September 1991.

5. Risk Assessment and Risk Tolerance

Risk tolerance was defined by Layfield (the Sizewell Public Inquiry, 1987) as follows:

* "...tolerance does not mean acceptability. It refers to the willingness to live with a risk to secure certain benefits, and in the confidence that it is being properly controlled. To tolerate a risk means that we do not regard it as negligible or something that we might ignore, but rather as something that we need to keep under review and to reduce still further if and as we can." *

The use of blood products is a good example of risk tolerance. The human immunodeficiency virus can be taken as an appropriate example mimicking hepatitis B and hepatitis C, all of which share a similar epidemiology.

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In January 1983 two articles were published in the prestigious New England Journal of Medicine and a Leading Article that the evidence that large pool Factor VIII concentrates contained an infective agent could not be ignored. The reason for the continued use of Factor VIII during 1983-84 was simply that there was a consensus that the benefits obtained from concentrate therapy greatly exceeded any harm that might ensue. At a meeting of the World Federation of Haemophilia in September 1984, there was a statement which "urged haemophiliacs not to withhold treatment for fear of AIDS. The risk of AIDS was 1:100 in antibody-positives". A 1984 Lancet Editorial ended "We must not forget that the commonest cause of haemophiliac death is bleeding".

The continued use of blood derivatives prepared from large pools of plasma can only be understood in terms of risk analysis i.e. that the perceived risks were far outweighed by the enormous benefits.

6. Opinion

1. Several hepatitis viruses and HIV constitute the main hazard of the transfusion of blood and certain blood derivatives prepared from large pools of plasma.
2. Paid donors constitute a particularly high-risk group for transmitting viral hepatitis.
3. The introduction of specific serological tests for screening blood for hepatitis B and C (and other bloodborne viruses) has increased considerably the safety of blood. However, the overwhelming evidence indicates that the elimination of paid professional donors has been the single most important factor in reducing the incidence of post-transfusion hepatitis.
4. Methods for inactivating residual virus in blood derivatives were difficult to develop.
5. The introduction of routine screening of all blood donors for hepatitis C in the UK on 1st September 1991 was not delayed unreasonably beyond the production, availability and secure supply of more specific and sensitive second generation tests and confirmatory tests.
6. Risk assessment and risk tolerance are significant factors in the use of blood and blood derivatives.

Signed.....

GRO-C

A.J. Zuckerman, MD, DSc, FRCP, FRCPATH
 Professor of Medical Microbiology in the University of London,
 Director of the WHO Collaborating Centre for
 Reference and Research on Viral Diseases,
 Member of the WHO Expert Panel on Viral Diseases, and
 Honorary Consultant in Microbiology and Clinical Virology

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Your ref:

Our ref: BGD/RL/S178.1

Date: 11/07/96

Reply to:

Edinburgh
Mr. Donald

Dear Professor Zuckerman

Solicitors' Hepatitis Group (Scotland)

I have now the comments of my various colleagues in respect of your report and would appreciate your further comments on the following points:-

1. The general flavour of your opinion and conclusion are to the effect that given all the circumstances and the relative risks, there is no realistic basis for a claim against either the Government or the Scottish National Blood Transfusion Service. In other words, given the circumstances prevailing in respect of the identification of the hepatitis C virus, it is likely that the "state of the art" defence would be capable of being established. Is that correct?
2. I appreciate the details you give in respect of the history of identification of the virus and the difficulties facing those attempting to establish an effective detection test. You say on page 5 of your report that preliminary trials were completed in the autumn of 1990 at which time two manufacturers were planning to introduce more sensitive and specific tests. These tests were in fact available in February 1991 but required evaluation and further testing in the U.K. so that they were not introduced universally here until 1st September 1991. Would it have been possible, and if so, appropriate (apart from resources), to have introduced the tests earlier even by only a few months?
3. I also note your comments with regard to risk assessment and risk tolerance as compared with the perceived risks of infection and that the use of blood and blood derivatives from large pools of plasma is largely beneficial for those sadly suffering from haemophilia. Would you not agree that the same considerations do not apply to matters of whole blood for transfusion purposes and that earlier effective screening and indeed surrogate testing would have been on balance more beneficial than the risk of transfusing infected blood? I say this because although the hepatitis C virus was not identified .../

Not known before this
identified until 1989, from 1985 the ~~Non-A Non B virus was known of~~. What if any action, whether by screening or testing, could have been taken against the transfer of this unidentified virus and its infective potential?

4. Equally I note that you say that the most important factor in reducing the instance of post-transfusion hepatitis has been the elimination of paid professional blood donors. Since as far as I know the U.K. has been self-sufficient in whole blood, certainly since the mid-1980s, is it the case do you think, that the infection has arisen purely because of donations by infected donors in the U.K. or was it the case that whole blood was being imported also?
5. Indeed was there no other source of blood products for import rather than the United States, bearing in mind the well-known practice there of paying blood donors?
6. You will recall of course that I also asked in my original instructions for you to comment on liability of the Government and SNBTS for patients infected with hepatitis B. There are very few cases, but do you agree that since there has been an effective test since 1985, anyone infected with hepatitis B is likely to suggest medical negligence?
7. One of my colleagues has asked the following question on which I would be glad of your comments. "Are you aware from the information available whether haemophiliacs were advised of the risks from blood derivatives prepared from large pools of plasma as compared with the risk of death from bleeding". You make reference at page 4 to the risk of contracting AIDS from blood products as being 1:100 but are there risk figures in respect of the contraction of hepatitis C and death through bleeding? I appreciate that you have given general views on the benefits to haemophiliacs but do you consider that they were given sufficient information or advice to enable them to make an informed choice, even if it were only in respect of the possible infection with the Non-A Non-B virus and its possible consequences?

I shall look forward to hearing from you.

Yours sincerely

BRIAN G DONALD

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GRO-C



17th July 1996

Your Ref: BGD/RL/S178.1

Mr Brian G. Donald,
J & A Hastie, Solicitors,
43 York Place,
Edinburgh EH1 3HT

Dear Mr Donald,

Thank you for your letter of 11th July 1996. I set out my comments in the order of the issues raised in your letter.

1. There is no realistic basis on scientific grounds, in my opinion, for a successful claim against either the Government or the SNBTS in respect of the identification of hepatitis C virus (HCV) and the introduction of screening tests for blood donors in the UK.
2. Please refer to item 4 and in particular to page 5 of my original report.

Although second generation tests became available in February 1991, the tests required laboratory evaluation. When the evaluation was completed, one manufacturer altered the test format so that all the tests had to be repeated in the Spring and early Summer of 1991. Two other ancillary factors should be noted:

- a. The heavy commitment of the Blood Transfusion Services in the national interest to the war effort in the Gulf in the Winter of 1991.
- b. The need to train staff at Transfusion Laboratories when new tests are introduced, the provision of equipment and securing an adequate and reliable supply of test kits and reagents.

I do not consider that it would have been realistic to introduce tests for HCV of all blood donors uniformly throughout the UK before 1st September 1991.

3. Surrogate tests do not offer specificity, which in my view is essential because of the potentially large number of false-positive and more importantly false-negative results. There are no confirmatory tests for surrogate markers. There were no tests for infectivity at the time.

.....Continued

Mr Brian G. Donald
17th July 1996

4. Fortunately, blood donations in the UK are entirely voluntary. However, self-sufficiency for the provision of blood products from voluntary sources has not been attained.

The European Union set out its target of achieving self-sufficiency in blood products in 1989. This has not been attained. In 1994, 60% of the plasma used in the European Union's plasma products was collected in the United States. The European Union has avoided so far, to my knowledge, setting a date for ending the importation of plasma products.

5. Manufactured blood products from countries outside North America and some producers in Europe may carry higher risks of infection. Much depends on Good Manufacturing Practices, donor selection and requirements set by the National Control Authorities. (Please refer also to Item 3 on Page 3 of my report).

6. It is incorrect to assume that medical negligence is the reason for hepatitis B infection by screened blood. There may be several technical reasons for inability to detect residual infectivity, and there are, in any case, no absolute tests in biological terms. The risk of hepatitis B today in screened blood in the USA, for example, is 1:50,000 units.

7. Patients with haemophilia are generally aware of the possible risk of viral infection and its consequences. ~~Note, for example, the statement on AIDS issued by the World Federation of Haemophilia in September 1984 (Page 6 of my report).~~ I cannot comment, however, on whether each patient was informed of the risk by each Haemophilia Centre in the country. ~~no?~~

~~In general terms, the risk of death from bleeding or from serious tissue damage as a result of bleeding is certainly more immediate than the consequences of infection with HCV where the disease may evolve slowly over a period of 20-30 years or longer. However, this is not my field of expertise and the question concerning bleeding should be asked of a physician specialising in coagulation disorders. You may wish to consult Dr Christine Lee, Director of the Haemophilia Centre at the Royal Free.~~

I hope that the above comments are helpful.

Yours sincerely,

GRO-C

Professor A.J. Zuckerman
Dean of the School of Medicine and
Director of the WHO Collaborating Centre for
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Your ref: Our ref: BGD/PMcC/S178.1 Date: 15 August, 1996 Reply to: Mr Donald
EDINBURGH

Dear Professor Zuckerman

Solicitors Hepatitis Group (Scotland)

I refer to your letter of 17th July with your further comments in respect of the matters raised in my letter of 11th July.

There are a number of other points arising on which I would appreciate your comments although I do take on board the point you make at the end of your letter of 17th July that you cannot comment on the risk of death from bleeding or tissue damage being more immediate than the consequences of infection with hepatitis C and that this more properly could be dealt with the other expert you recommend. Nevertheless I would raise the following matters for your comments.

Hepatitis B Infection

1. I note your comments in item 6 of your letter of 17th July as to the fact that there are no absolute tests in biological terms and that there may be several technical reasons for inability to detect residual infectivity. You mention statistics in the USA which show a risk of hepatitis B resulting even from screened blood in 1/50,000 units. Are there statistics for the UK bearing in mind that effective screening tests were introduced in 1985?
2. Could you also explain what technical reasons there are for the inability to detect residual infectivity.

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Your ref: BGD/PMcC/s178.1

4 September 1996

Mr Brian G Donald
J & A Hastie, Solicitors
43 York Place
Edinburgh EH1 3HT

Dear Mr Donald

Solicitors Hepatitis Group (Scotland)

Thank you for your letter of 15 August, 1996 which I found on my return from leave. The following are my comments on the further points raised by members of the Solicitors Hepatitis Group (Scotland).

1. There are no published absolute figures in the UK for hepatitis B associated with the transfusion of screened blood, but based on cases reported to Regional Transfusion Centres it was estimated in 1993 that there are probably 100 infections per annum.
2. As a matter of principle, all laboratory tests should be conducted by nationally approved methods, or they must be carried out by validated methods giving equivalent results. A minimum set of controls are included with every series of tests. Reagents used in tests conform to national and international standards. Tests carried out by the National Blood Transfusion Services meet these criteria.

There are many technical reasons why biological tests are not absolute and may not detect infectivity in all cases. These include the following:

Continued ...

- The "window phase". This is the period between infection and the detectability of virological markers of infection. An example is an infectious blood donor who is in the early incubation period of acute hepatitis B. Over the years much effort has been devoted, with success, to shortening significantly the "window" phase.
- Hepatitis B surface antigen variants and mutants, which may not be detectable by current tests particularly those tests based on highly specific monoclonal antibody assays.
- Limits to the sensitivity of screening tests, which are generally based on enzyme immunoabsorbent assays.
- Circulating antigen-antibody complexes so that free viral antigen is not available to the detector system.

The above are some of the reasons why consideration is being given to the introduction of nucleic acid based techniques and the need to improve further the sensitivity of screening tests.

3. Ministers responsible for the Departments of Health (in England, Wales, Scotland and Northern Ireland) receive advice of course from the Chief Medical Officers through various expert advisory committees, for example the Hepatitis Advisory Group, the Advisory Committee on the Microbiological Safety of Blood and Tissues, the Microbiology Advisory Committee, the National Blood Authority and its various advisory committees and the committees of the Regional Blood Transfusion Centres, the Expert Advisory Committee on AIDS and others. There is therefore no lack of expertise and information. Guidelines are published by the Department of Health from time to time and these are widely distributed.

Information about risks associated with a particular treatment falls within the domain of the clinician responsible for the patient.

4. * The existence of non-A, non-B hepatitis was known to all national Health Authorities in 1981-1985. *

5. Please refer to my comments above under item 3.

* You should also be aware that as long ago as 1976, Granada Television transmitted two well-documented World in Action programmes on public television (Channel 3) on *

Continued ...

- 3 -

hepatitis risks associated with commercial blood and blood products under the title "Blood Money". These programmes were televised subsequently in many countries throughout the world including the USA. A further World in Action programme, "The Blood Business", was transmitted in 1980. I believe, therefore, that the lay public will be aware of the risks associated with blood transfusion.

Although I participated in the preparation of these programmes, I regret that I do not have copies of the tapes. The topic has also been aired in the popular press from time to time.

I attach a note of a nominal fee for the work carried out on 17 July, 1996 and that associated with the above annotations.

Yours sincerely

GRO-C

Professor A J Zuckerman
Director of the WHO Centre
and
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Date: 24th September 1996

Reply to:

Brian G Donald
Edinburgh

Dear Professor Zuckerman

Solicitors' Hepatitis Group (Scotland)

I thank you for your letter of 4th September and have now had an opportunity of considering your further comments. I will arrange for settlement for your further fee as soon as possible since I did not anticipate this in that all the question I have put arise from your original report. I do however accept that you have been involved in further work and will arrange for settlement as soon as possible.

Meanwhile however I trust you would be prepared to clear up two small points arising from your current letter.

1. You say that in 1993 it was estimated that there were probably 100 hepatitis B infections in the UK associated with the transfusion of screened blood. Is it possible for you to relate this to the number of transfusions of screened blood per annum since of course the statistics in the USA which you mentioned in your letter of 17th July were so related.
2. Given your further comments on the reasons for why biological tests are not absolute, is it the case that there will never be an absolute test and does that apply to the hepatitis C test which was introduced in September 1991 in the UK? If so, can you say approximately per annum how many persons continue to be infected with the hepatitis C virus from screened blood?
3. Finally, you say that consideration is being given to the introduction of nucleic acid-based techniques for the improvement of the sensitivity of screening tests. Are such techniques used in other parts of the world and in particular the USA? If so, when were they introduced?

I shall be glad of your further comments in these matters and am much obliged for your continued assistance.

Yours sincerely

Brian G Donald

St George's Hospital School of Medicine
UNIVERSITY OF LONDON

World Health Organisation Collaborating Centre
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GRO-C

1st October 1996

Your Ref: BGD/RL/S178.1

Mr Brian G. Donald,
J & A Hastie Solicitors,
43 York Place,
Edinburgh EH1 3HT

Dear Mr Donald,

I respond briefly to your letter of 24th September 1996 if for no other reason than to limit the amount of time I spend on this matter and to contain the professional costs.

1. The following figures were provided to me by the North London Blood Transfusion Centre.

Last year, the Blood Transfusion Services in the UK collected approximately 2,300,000 units of blood and issued 2,000,000 units. Approximately 800,000 patients were transfused. Other annual figures will have to be sought from the National Blood Authority.

2. A study in the USA in 1992 assessed the efficacy of anti-HCV screening based on the measurement of anti-HCV in a pre-transfusion and a 6-month post-transfusion sample. This study involved 25,832 units administered to 2,415 patients. The residual risk of transfusion-associated hepatitis was 1 in 3,333 units transfused. (in 286 patients?)
3. A revised mathematical risk estimate, as another approach, calculates for the USA the current risk of hepatitis C transmission by screened blood as 1 in 103,000 (95% confidence interval 28,000 to 288,000), and of hepatitis B transmission 1 in 63,000 (95% confidence interval 31,000 to 147,000).

Risk was estimated on a calculation based on the length of the window period before the detection of a marker of infection and on the measured incidence of new infections in the donor population as the product of the window period x the incidence of new infection.

I do not have comparable estimated figures for the UK.

.....Continued



Mr Brian G. Donald
1st October 1996

3. I do not consider it likely that absolute biological tests for hepatitis viruses will become available in the foreseeable future.
4. Nucleic acid-based tests are not used routinely for screening blood donors in the USA or indeed elsewhere, to my knowledge.

Yours sincerely,

GRO-C

Professor A.J. Zuckerman
Director of the WHO Centre and
Dean of the School of Medicine